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METHODS

The Implanted Defibrillator: Relation of Defibrillating Lead Configuration and Clinical Variables to Defibrillation Threshold

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Forty-two defibrillating lead systems for the automatic implantable defibrillator were implanted and tested in 41 patients. Two basic lead configurations were used: 1) spring-patch, consisting of a transvenous superior vena cava spring electrode as the anode and an apical or left lateral ventricular patch electrode (either small [13.9 cm^2] or large [27.9 cm^2]) as the cathode; and 2) patch-patch, consisting of an anterior right ventricular patch as the anode and a posterior left ventricular patch as the cathode. Of the 42 lead systems, 10 were spring-patch and 32 were patch-patch combinations. The defibrillation threshold for the patch-patch combinations (9.8 ± 6.5 J, mean ± standard deviation) was significantly ($p < 0.01$) lower than that for the spring-patch combinations (19.1 ± 10.3 J). Subgroup analysis revealed the lowest defibrillation thresholds for patch-patch combinations with at least one large patch. Total surface area of defibrillating leads was strongly negatively correlated with the defibrillation threshold ($p < 0.005$).

Analysis of the relation of clinical variables to defibrillation threshold revealed that only amiodarone therapy was independently associated with a significantly ($p < 0.05$) higher defibrillation threshold. Thus, surface area of the defibrillating leads is a critical determinant of the defibrillation threshold for the implanted defibrillator. Patch-patch lead systems with at least one large patch may provide an increased safety margin for defibrillation. Conversely, amiodarone therapy is associated with higher defibrillation thresholds and may decrease the margin of safety.

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The automatic implantable defibrillator (AID-B, Intec Systems) is a device capable of automatically identifying and correcting ventricular tachycardia and fibrillation (1–3) and has been shown to significantly increase survival in patients at high risk for recurrent cardiac arrest (4). The current device is a result of an evolutionary development process begun by investigators over a decade ago (5–8).

The defibrillating lead system of the automatic implantable defibrillator in the majority of human implants has consisted of a transvenous spring electrode (a titanium coil with a surface area of 6.5 cm^2; personal communication, S. M. Bach, Jr., MD, Intec Systems) serving as the anode, and an apical or left lateral ventricular patch electrode (a titanium mesh flat electrode with a surface area of either 13.9 or 27.9 cm^2) serving as the cathode (spring-patch configuration).

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It is also possible to use two patch electrodes for defibrillation (patch-patch configuration). This configuration is obtained by placing one patch posteriorly or posterolaterally on the left ventricle, with the second patch positioned anteriorly over the right ventricle. Two small patches, two large patches or one of each size are possible options for the patch-patch configuration.

It has been reported by Winkle et al. (9) that when reliable defibrillation is not obtainable with 25 to 30 J (the output of the currently used automatic implantable defibrillator generator) using the spring-patch configuration, the patch-patch configuration may result in lower defibrillation thresholds. This report summarizes our experience with various defibrillating lead configurations in the first 41 patients undergoing lead implantation at our institution.

Methods

Study patients. Forty-one consecutive patients who had the lead system of the automatic implantable defibrillator implanted were studied. One patient had two lead system implants because infection necessitated removal of the first system. The defibrillating lead configuration and patient

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characteristics are shown in Table 1. Thirty-two of the 41 patients were male and 9 were female, ranging in age from 13 to 81 years (mean 57.4).

Thirty-two of the patients had experienced an episode of cardiac arrest and nine had symptomatic recurrent sustained hypotensive ventricular tachycardia. Thirty-six patients had coronary artery disease, three patients had nonischemic congestive cardiomyopathy, one patient had hypertrophic nonobstructive cardiomyopathy and one patient had no detectable structural heart disease. The mean left ventricular ejection fraction as measured by radionuclide or contrast ventriculography was 35.8% (range 13 to 85).

Operative procedure. General anesthesia was induced with a combination of morphine and benzodiazepine. Maintenance anesthesia was provided by a combination of nitrous oxide and a narcotic (usually fentanyl). The lead system

Table 1. Patient Data Including Defibrillation Threshold and Defibrillating Lead Configuration in 42 Patients

Case	Age (yr) & Sex	Diagnosis	LVEF (%)	Arrhythmic Event	Amiodarone	Digoxin	Type I Drug	Other Cardiac Surgery	DFT	Defibrillating Lead Configuration
1	61M	CCM	20	CA	+	+	+	None	20	S_P-P_S
2	51M	CAD	27	CA	+	+	−	An, CABG	20	S_P-P_S
3	35F	CAD	45	VT	+	+	+	CABG	40	S_P-P_S
4*	49M	CAD	17	CA	+	+	−	None	15	S_P-P_L
5	58M	CAD	25	CA	+	+	−	CABG	25	S_P-P_L
6	58M	CAD	34	CA	+	+	−	An	10	S_P-P_L
7	68M	CAD	23	VT	−	+	+	CABG	15	S_P-P_L
8	30M	HCM	85	CA	+	−	−	None	25	S_P-P_L
9	50M	CAD	50	CA	−	−	+	An, CABG	20	S_P-P_L
10	50M	CAD	26	VT	−	+	−	An, CABG	1	S_P-P_L
11	72F	CAD	27	CA	−	+	+	None	15	P_S-P_S
12	13F	PED	71	CA	−	−	+	None	15	P_S-P_S
13	48M	CAD	69	CA	+	+	−	None	15	P_S-P_S
14	57F	CAD	37	CA	+	+	−	None	10	P_s-P_S
15	49M	CAD	56	CA	−	−	−	CABG	10	P_S-P_S
16	49F	CAD	46	CA	−	+	−	An	2	P_S-P_L
17	57M	CAD	33	CA	−	−	+	An, CABG	5	P_S-P_L
18	58M	CAD	24	CA	+	+	+	None	20	P_S-P_L
19	71M	CAD	32	CA	+	+	+	CABG	5	P_S-P_L
20	68M	CAD	34	CA	+	+	+	An, CABG	15	P_S-P_L
21	64M	CAD	40	VT	−	−	−	An, CABG	5	P_S-P_L
22	51F	CAD	29	CA	−	−	−	CABG	1	P_S-P_L
23	81M	CAD	47	CA	−	−	+	CABG	5	P_S-P_L
24	57M	CAD	25	VT	−	+	+	An, CABG	10	P_S-P_L
25	65M	CAD	13	CA	+	+	+	An, CABG	1	P_S-P_L
26	48M	CAD	26	VT	−	−	+	CABG	20	P_S-P_L
27	66M	ASD, CAD	29	CA	+	+	+	An, ASDR, CABG	30	P_S-P_L
28*	49M	CAD	17	CA	+	+	+	An, CABG	15	P_L-P_L
29	59M	CAD	30	CA	+	+	+	An, CABG	5	P_L-P_L
30	62F	CAD	49	CA	+	−	+	CABG	2	P_L-P_L
31	54M	CAD	9	CA	−	+	+	None	10	P_L-P_L
32	72M	CAD	24	CA	+	+	+	CABG	5	P_L-P_L
33	70F	CAD	14	CA	+	+	−	None	10	P_L-P_L
34	40M	CAD	42	CA	−	−	+	An, CABG	5	P_L-P_L
35	48F	CCM	30	CA	−	+	−	None	20	P_L-P_L
36	76M	CAD	31	VT	−	+	+	CABG	5	P_L-P_L
37	76M	CAD	82	CA	−	−	−	CABG	5	P_L-P_L
38	58M	CAD	36	VT	−	+	+	None	1	P_L-P_L
39	64M	CCM	32	CA	−	−	−	None	10	P_L-P_L
40	69M	CAD	40	CA	+	+	+	CABG	10	P_L-P_L
41	59M	CAD	51	CA	−	−	−	CABG	10	P_L-P_L
42	60M	CAD	27	CA	+	−	+	An, CABG	15	P_L-P_L

*Patients 4 and 28 are the same patient in whom two different lead systems were implanted because of infection of the first leads. An = ventriculotomy with subendocardial resection or cryoablation, or both; ASD = atrial septal defect; ASDR = atrial septal defect repair; CA = cardiac arrest; CABG = coronary artery bypass grafting; CAD = coronary artery disease; CCM = congestive cardiomyopathy; DFT = defibrillation threshold; F = female; HCM = hypertrophic cardiomyopathy; LVEF = left ventricular ejection fraction; M = male; PED = primary electrical disease; P_L = large patch electrode (27.9 cm^2); P_S = small patch electrode (13.9 cm^2); S_P = spring electrode; VT = sustained ventricular tachycardia; + = taking the drug or drugs; − = not taking the drug or drugs.

was placed by means of a left anterior thoracotomy in 12 patients and a median sternotomy in 30 patients. Of the latter 30 patients, 29 underwent a surgical procedure in addition to lead system placement. Coronary artery bypass grafting was performed in 15 patients, bypass grafting in conjunction with left ventriculotomy (with subendocardial resection or cryoablation, or both) in 12 patients (1 of whom also underwent repair of a congenital atrial septal defect) and left ventriculotomy alone (with subendocardial resection or cryoablation, or both) in 2 patients. When other cardiac operations were performed, the automatic implantable defibrillator leads were installed and tested after these procedures had been completed.

Electrode positioning. In all instances, the placement of the electrodes was designed to include the largest mass of myocardium possible between them. The initial electrode configuration was determined by the operating surgeon on the basis of his intraoperative assessment. During the first 10 implants, a spring-patch configuration was utilized and not altered unless unacceptably high defibrillation thresholds were obtained. During subsequent implants (partially due to experience with difficulty in spring electrode placement and migration of several spring electrodes out of position), a two patch configuration was elected, again with the size of the implanted patches being chosen by the operating surgeon. A number of factors influenced the decision regarding choosing the patch size, including the location of any aortocoronary vein grafts, internal mammary artery grafts or ventriculotomy and overall cardiac size.

When utilized, the spring electrode (the anode of the shocking lead system) was introduced through a 14F introducer placed by means of a percutaneous puncture of one of the subclavian veins and advanced under fluoroscopic guidance to the mid right atrium. In three cases, the spring electrode was introduced through the innominate vein when technical factors precluded passage from either subclavian approach. The patch electrode (cathode of the shocking lead system) for the spring-patch configuration was then placed over the apical or lateral left ventricle and secured by suturing its edges to the ventricular epicardium. When two patches were utilized (patch-patch configuration), one was sutured to the anterior right ventricular surface (and served as the anode) and the other was sutured to the posterior or posterolateral left ventricular epicardium (and served as the cathode). Two epicardial screw-in electrodes (model K-54, Intec Systems) were employed to record electrograms for rate counting and synchronization of shocks. In one patient, an endocardial rate counting lead was used because of inadequate electrographic recordings from the epicardial surface.

Defibrillation threshold testing. Intraoperative defibrillation threshold testing was performed after the lead system was installed utilizing an external cardioverter defibrillator (Intec Systems) that delivers a truncated exponential shock waveform (identical to the waveform of the automatic implantable defibrillator pulse generator), with adjustable delivered energies from 1 to 40 J (in increments of 1 J at energy levels between 1 and 5 J and increments of 5 J at energy levels between 5 and 40 J). Ventricular fibrillation was induced by brief epicardial application of 60 Hz, 7.2 V (root mean square) alternating current. The shocks were delivered after 5 to 10 seconds of ventricular fibrillation. Ventricular fibrillation was defined as a ventricular tachyarrhythmia during which distinct QRS complexes could not be discerned in the surface electrocardiographic leads (I, aVF and V_6).

The initial energy level tested was 20 J. If successful, decrements of 5 J were tried until the 5 J level was reached or defibrillation was unsuccessful. If 5 J was successful, a 1 J shock was then tested. All unsuccessful attempts were followed immediately by a 40 J "rescue" shock, so that only one energy level was tested for each episode of ventricular fibrillation. Defibrillation trials were separated from one another by intervals of 1 minute. If the initial 20 J shock was unsuccessful, increments of 5 J were tested until defibrillation was achieved. The first unsuccessful shock and the lowest successful energy level were then repeated once. The defibrillating threshold was defined as the lowest energy that terminated ventricular fibrillation.

Statistical analysis. Unpaired *t* tests and the one-way analysis of variance (10) were used to evaluate the effects of several factors on the defibrillation threshold. These factors were gender, diagnosis, presenting arrhythmia, concomitant cardiovascular surgery and treatment with amiodarone, digoxin or one or more type I antiarrhythmic drugs (as defined by Vaughan Williams [11]). Pearson's correlation was used to evaluate the effects of age, surface area of defibrillating leads (total exposed titanium surface of the anode plus the cathode) and left ventricular ejection fraction (12). Multiple linear regression was then used to extract those variables providing independent information regarding the defibrillation threshold. All data are expressed as mean values ± 1 SD.

Results

Relation of lead configuration to defibrillation threshold. Of the 42 lead systems, 10 were spring-patch (Fig. 1) and 32 were patch-patch (Fig. 2) combinations. The mean defibrillation threshold for all of the patch-patch combinations (9.8 ± 6.5 J) was significantly ($p < 0.01$) lower than that for the spring-patch configuration (19.1 ± 10.3 J). Subgroup analysis based on lead configuration, patch size and surface area of the defibrillating leads is shown in Figure 3. The total surface area of the shocking leads was strongly negatively correlated with the defibrillation threshold ($p < 0.005$); the larger exposed lead areas yielded the lowest defibrillation thresholds. The lowest thresholds were ob-

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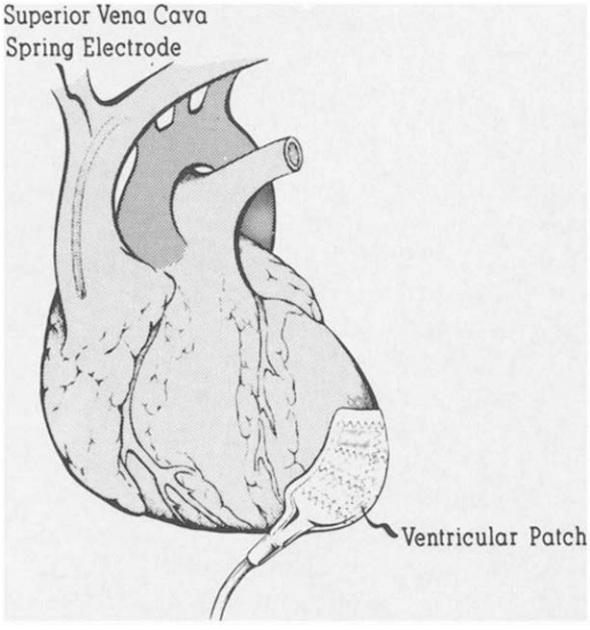


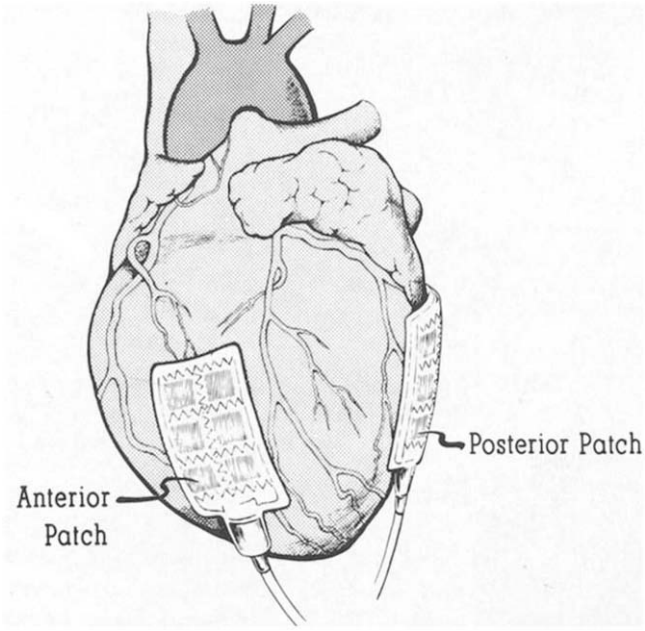
Figure 1. The spring-patch defibrillating lead configuration. A transvenous spring electrode (anode) in the superior vena cava and an apical patch electrode (cathode) on the anterior surface of the left ventricle are utilized for defibrillation.

tained with patch-patch configurations having at least one large patch.

Influence of clinical variables on defibrillation threshold (Table 2). Amiodarone therapy was associated with a significantly ($p < 0.05$) higher defibrillation threshold. Although patients with cardiomyopathy and those taking digoxin had a higher mean defibrillation threshold, this difference did not reach statistical significance. Similarly, age, presenting arrhythmic event, use of a type I antiarrhythmic drug and whether additional cardiac surgery was performed had no statistically significant effect on defibrillation threshold. The lower mean defibrillation threshold for the group having aneurysmectomy alone was not significantly different from the mean defibrillation threshold for patients having no additional cardiac surgery. There was also no relation between defibrillation threshold and left ventricular ejection fraction ($r = 0.022$, $p = NS$). Age was negatively correlated with defibrillation threshold ($r = -0.364$, $p < 0.01$).

Multivariate analysis. When the variables were combined in a multivariate analysis, the surface area of the defibrillating leads was most strongly associated with defibrillation threshold ($p < 0.01$). Amiodarone therapy also continued to contribute a significant ($p < 0.05$) effect on the defibrillation threshold. Patient age did not contribute any significant additional independent information in this analysis.

Figure 2. The patch-patch defibrillating lead configuration. An anterior right ventricular patch electrode (anode) is paired with a posterior left ventricular patch electrode (cathode) for defibrillation.



Discussion

Lead configuration and defibrillation threshold. The implanted defibrillator was initially conceived as a totally transvenous device utilizing a catheter electrode system (13). Subsequently, it was shown in dogs that the most consistent and lowest energy requirements for defibrillation were achieved with patch electrodes placed on the base and apex of the heart (6,14). A superior vena cava spring electrode with an apical patch electrode was found to be almost as effective and functioned well in a chronic dog model (15), and was then adopted for the initial human implants (1).

Our data indicate that the surface area of the defibrillating leads is one critical determinant of the energy required to achieve defibrillation. Defibrillation relates to the delivery of a current density adequate to depolarize a critical mass of myocardium (16). In studies of transthoracic defibrillation, it has been shown in dogs that larger electrode paddles improved the success rate of ventricular defibrillation (17), possibly related to a lower transthoracic impedance with larger electrode paddles (18,19). Electrode surface area may become excessive, however, resulting in more extracardiac current flow, with inadequate current density delivered to the heart for defibrillation (20). An analogous situation may well exist with implanted defibrillating electrode systems. Patch systems that are excessively large may lead to "shunting" of current between the edges of the electrodes instead of through the intervening myocardium. Optimal electrode size for the implanted shocking leads has not been defined and, in fact, will probably need to be individualized. As a practical matter, because the automatic implantable defibrillator has a finite delivered energy in the range of 25 to

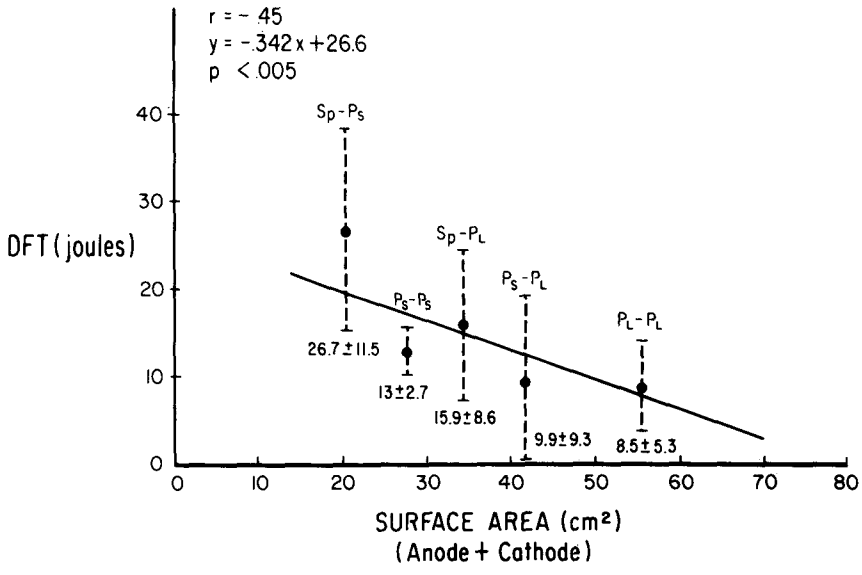


Figure 3. Relation of defibrillation threshold (DFT) to the surface area of the defibrillating leads (exposed titanium of the anode plus cathode). Data points (expressed as mean ± 1 standard deviation) are shown for each lead configuration and expressed numerically below. The **solid line** ($y = -0.342x + 26.6$) is the least squares line for the data points. P_L = large patch electrode (27.9 cm^2); P_S = small patch electrode (13.9 cm^2); S_P = spring electrode.

35 J, factors that reduce the threshold for defibrillation will increase the margin of safety. Our data strongly suggest that one means of reducing the defibrillation energy requirement is the implantation of a two patch electrode system and that, preferably, the system should include at least one large patch.

Effect of antiarrhythmic agents on defibrillation threshold. Amiodarone therapy was associated with a significantly higher defibrillation threshold independent of the surface area of the defibrillating leads or any other variable analyzed. Therefore, if a patient with an implanted defibrillator is started on amiodarone therapy, repeat testing is mandatory to ensure that the device is still capable of achieving defibrillation. Conversely, if a patient already taking amiodarone has a relatively high defibrillation threshold, discontinuing the drug may lower the threshold to a more acceptable level. Amiodarone-induced refractoriness to cardioversion (using the implanted defibrillator lead system) has been reported by Fogoros (21) in a patient in whom a 40 J shock delivered through the implanted leads was unsuccessful in terminating ventricular fibrillation while the patient was receiving amiodarone therapy. After discontinuation of amiodarone for 3 months, defibrillation was accomplished with 10 J.

The role of other antiarrhythmic agents in this context is not well defined. In the current study, no significant relation was found between either digoxin therapy or type I antiarrhythmic drug therapy and defibrillation threshold (al-

Table 2. Influence of Clinical Variables on Defibrillation Threshold

Variable		Mean DFT	p Value
Sex	M	11.9	NS
	F	12.8	
Diagnosis	CAD	10.8	NS
	CM	18.8	
Arrhythmic event	CA	12.3	NS
	VT	11.3	
Other cardiac surgery	None	14.3	NS
	An	6.0	
	None	14.3	NS
	CABG	11.3	
	None	14.3	NS
	An, CABG	10.2	
Amiodarone	(+)	15.1	< 0.05
	(−)	9.0	
Digoxin	(+)	13.7	NS
	(−)	10.2	
Type I drug	(+)	12.6	NS
	(−)	11.4	

CM = cardiomyopathy; NS = not significant; other abbreviations as in Table 1.

though it should be noted that the statistical analysis did not address individual type I antiarrhythmic agents due to the multiplicity of single agents employed). This is in agreement with data from animal experiments (22) suggesting that there is no acute change in defibrillation threshold with digoxin or procainamide (administered by intravenous bolus) using the automatic implantable defibrillator lead system. However, several type I antiarrhythmic drugs (lidocaine, phenytoin and quinidine) have been reported (23,24) to raise transthoracic ventricular defibrillation thresholds in dogs. In contrast, bretylium and clofilium have been shown to decrease the canine transthoracic ventricular defibrillation threshold (25), an effect with potential benefit in patients with implanted defibrillators. The effects of antiarrhythmic and other drugs on defibrillation thresholds with implanted lead systems is in need of further investigation.

Limitations of the study. The clinical determination of the defibrillation threshold as a single quantity of energy, implying a sharp distinction between effective and ineffective energy levels, is probably a misleading concept. It has been shown in dogs that there is a range of energies that are more or less successful in producing defibrillation (26). During our initial implantation experience, it was not clear how well defibrillation threshold testing would be tolerated by patients. Therefore, we elected to minimize the number of fibrillation-defibrillation trials. We have, however, noted no untoward effects from the defibrillation trials. Similarly, it would have been informative to test every lead configuration in every patient (which would have allowed the patient to serve as his or her own control). Once a satisfactory lead configuration was found, however, we elected not to conduct trials of other lead configurations to minimize the potential for patient morbidity. Additionally, it should be noted that random selection of energies for the defibrillation trials might have given additional information regarding the effect of time and previous shocks on the efficacy of subsequent shocks since in our present protocol, lower energy shocks were delivered only after successful higher energy shocks.

The data reported are specific to the lead systems and the truncated exponential waveform utilized by the Intec device. Extrapolation of these results to other defibrillation waveforms is not possible.

Conclusions. There is a significant relation between defibrillating lead configuration and defibrillation threshold for the automatic implantable defibrillator. Specifically, it appears that lead surface area is a critical determinant. Patch-patch lead systems with at least one large patch were associated with the lowest defibrillation thresholds and may provide a larger margin of safety for defibrillation. Additionally, amiodarone therapy was associated with significantly higher defibrillation thresholds. Therefore, defibrillation testing should be repeated if amiodarone therapy is initiated after defibrillator implantation to ensure continued efficacy. Because the time course of amiodarone effect on defibrillation threshold is unknown, the timing of repeat testing is empiric and we select an arbitrary time 4 to 6 weeks after initiation of the drug. Recommendations regarding other antiarrhythmic agents await further delineation of their role in modulating the defibrillation threshold.

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